What is claimed is:

1. A method of treating granulocytopenia in a subject, comprising administering to the subject an agent that inhibits the degradation of GM-CSF mRNA, thereby treating granulocytopenia in the subject.

2. The method of claim 1, wherein the granulocytopenia is relative.

3. The method of claim 1, wherein the granulocytopenia is absolute.

4. The method of claim 1, wherein the degradation of GM-CSF mRNA is inhibited by inhibiting the mRNA degradative activity of TTP.

5. The method of claim 1, wherein the agent that inhibits the degradative activity of TTP is a competitor of TTP.

6. The method of claim 5, wherein the competitor competes with TTP for binding on the AU-rich element (ARE) of GM-CSF mRNA.

7. The method of claim 5, wherein the competitor competes with TTP for binding on an mRNA degradative enzyme.

8. The method of claim 1, wherein the degradation of GM-CSF mRNA is inhibited by inhibiting the mRNA degradative activity of ERF1.

9. The method of claim 8, wherein the agent that inhibits the mRNA degradative activity of ERF1 is a competitor of ERF1.

10. The method of claim 9, wherein the competitor competes with ERF1 for binding on the AU-rich element (ARE) of GM-CSF mRNA.

11. The method of claim 9, wherein the competitor competes with ERF1 for binding on an mRNA degradative enzyme.

12. The method of claim 1, wherein the degradation of GM-CSF mRNA is inhibited by inhibiting the mRNA degradative activity of ERF2.

13. The method of claim 12, wherein the agent that inhibits the mRNA degradative activity of ERF2 is a competitor of ERF2.

14. The method of claim 13, wherein the competitor competes with ERF2 for binding on the AU-rich element (ARE) of GM-CSF mRNA.

15. The method of claim 13, wherein the competitor competes with ERF2 for binding on an mRNA degradative enzyme.

16. A method of treating granulocytopenia in a subject, comprising administering to the subject a mutant TTP that has reduced activity compared to wild type TTP.

17. The method of claim 16, wherein the activity of TTP reduced is degradation of GM-CSF mRNA.

18. The method of claim 17, wherein the mutant TTP is administered by delivering to the subject a nucleic acid that encodes the mutant TTP and allows expression of the mutant TTP in cells of the subject.

19. A method of treating granulocytopenia in a subject, comprising administering to the subject a mutant ERF1 that has reduced activity compared to wild type TTP.

20. The method of claim 19, wherein the activity of ERF1 reduced is degradation of GM-CSF mRNA.

21. The method of claim 19, wherein the mutant ERF1 is administered by delivering to the subject a nucleic acid that encodes the mutant ERF1 and allows expression of the mutant ERF1 in cells of the subject.

22. A method of treating granulocytopenia in a subject, comprising administering to the subject a mutant ERF2 that has reduced activity compared to wild type TTP.

23. The method of claim 22, wherein the activity of ERF2 reduced is degradation of GM-CSF mRNA.

24. The method of claim 22, wherein the mutant ERF2 is administered by delivering to the subject a nucleic acid that encodes the mutant ERF2 and allows expression of the mutant ERF2 in cells of the subject.

25. A mutant TTP that has a reduced TTP activity compared to wild type TTP.

26. The mutant TTP of claim 25, wherein the activity of TTP reduced is TTP binding to the ARE of GM-CSF mRNA.

27. The mutant TTP of claim 25, wherein the activity of TTP reduced is TTP binding to an mRNA degradative enzyme.

28. The mutant TTP of claim 25, wherein the activity of TTP reduced is degradation of GM-CSF mRNA.

29. The mutant TTP of claim 25, wherein the mutant is C124R.

30. The mutant TTP of claim 25, wherein the mutant is C147R.

31. A mutant ERF1 that has a reduced ERF1 activity compared to wild type ERF1.

32. The mutant ERF1 of claim 31, wherein the activity of ERF1 reduced is ERF1 binding to the ARE of GM-CSF mRNA.

33. The mutant ERF1 of claim 31, wherein the activity of ERF1 reduced is ERF1 binding to an mRNA degradative enzyme.

34. The mutant ERF1 of claim 31, wherein the activity of ERF1 reduced is degradation of GM-CSF mRNA.

35. A mutant ERF2 that has a reduced ERF2 activity compared to wild type ERF2.

36. The mutant ERF2 of claim 35, wherein the activity of ERF2 reduced is ERF2 binding to the ARE of GM-CSF mRNA.

37. The mutant ERF2 of claim 35, wherein the activity of ERF2 reduced is ERF2 binding to an mRNA degradative enzyme.

38. The mutant ERF2 of claim 35, wherein the activity of ERF2 reduced is degradation of GM-CSF mRNA.

39. A method of screening an agent for the ability to inhibit an activity of TTP, comprising the steps of:

a) cotransfecting a cell with a nucleic acid that encodes TTP and a nucleic acid that comprises an ARE downstream of a nucleic acid sequence encoding a reporter protein;

b) contacting the cell of step a) with the agent; and

c) comparing the expression of the reporter protein in the cell of step b) to the cell of step a) in the absence of the agent, an increase in reporter gene expression in the cells of step b) compared to the cells of step a) indicating that the agent has the ability to inhibit an activity of TTP.

40. A method of screening an agent for the ability to compete with TTP for binding to the ARE of mRNA, comprising the steps of:

a) transfecting a cell with a nucleic acid that encodes TTP;

b) obtaining a cytosolic extract of the cell of step a);

c) contacting the cytosolic extract of step b) with the agent;

d) contacting the cytosolic extract of steps b) and c) with a probe comprising an ARE;

e) comparing the binding of the probe to TTP in the cytosolic extract of step b) with the binding of the probe to TTP in the cytosolic extract of step c), the presence of reduced binding of the probe to TTP in the cytosolic extract of step c) indicating an agent that can compete with TTP for binding to the ARE of mRNA.

41. A method of stimulating the degradation of an mRNA molecule having an AU-rich element (ARE), comprising contacting the mRNA molecule with a tandem zinc finger (TZF) polypeptide consisting essentially of the tristetraprolin (TTP) zinc finger domain or comprising a TTP-like zinc finger domain, thereby stimulating degradation of the mRNA molecule.

42. The method of claim 41, wherein the TTP-like zinc finger domain is selected from the ERF1 zinc finger domain, the ERF2 zinc finger domain, and the XC3H-4 zinc finger domain.

43. The method of claim. 41, wherein the TZF polypeptide is selected from ERF1, ERF2, and XC3H-4.

44. ~~43~~. The method of claim 41, wherein the mRNA molecule is within a cytosolic extract.

45. ~~44~~. The method of claim 41, wherein the mRNA molecule is within a cell.

46. ~~45~~. The method of claim 41, wherein the mRNA molecule is within a patient or subject.

47. ~~46~~. The method of claim 41, wherein production of a polypeptide encoded by the mRNA molecule is decreased.

48. ~~47~~. The method of claim. 41, wherein the mRNA molecule encodes TNF-α.

49. ~~48~~. The method of claim. 46, wherein the polypeptide is TNF-α.

50. ~~49~~. The method of claim. 41, wherein the TZF polypeptide is administered to a patient or subject to treat, inhibit, or prevent a TNF-α-related disease or condition in the patient or subject.

51. ~~50~~. The method of claim. 41, wherein a nucleic acid encoding the TZF polypeptide is administered to a patient or subject to treat, inhibit, or prevent a TNF-α-related disease or condition in the patient or subject.

52 ~~51~~. The method of claim. 41, wherein the ARE is a class II ARE.

53 ~~52~~. A method of identifying a compound that modulates the activity of TTP or a TTP-like polypeptide, comprising:

a) contacting a sample with the compound, and

b) detecting or measuring the binding between an ARE and a TZF polypeptide consisting essentially of a TTP zinc finger domain or a polypeptide comprising a TTP-like zinc finger domain in the sample, whereby an increase or decrease in the binding between the ARE and the polypeptide, relative to the binding between the ARE and the polypeptide in the sample not contacted with the compound, identifies a compound that modulates the activity of TTP or a TTP-like polypeptide.

54 ~~53~~. The method of claim 52, whereby an increase in the binding between the ARE and the polypeptide identifies a compound that stimulates the activity of a TTP or a TTP-like polypeptide.

55 ~~54~~. The method of claim 52, wherein the method identifies a compound that stimulates degradation of an mRNA molecule comprising an ARE.

56 ~~55~~. The method of claim. 52, wherein the mRNA molecule encodes TNF-α.

57 ~~56~~. The method of claim 52, whereby a decrease in the binding between the ARE and the TZF polypeptide identifies a compound that inhibits the activity of TTP or a TTP-like polypeptide.

58 ~~57~~. The method of claim 52, wherein the method identifies a compound that inhibits degradation of an mRNA molecule comprising an ARE.

59 ~~58~~. The method of claim. 52, wherein the mRNA molecule encodes GM-CSF or IL-3.

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59. The method of claim 52, further comprising contacting the sample with an inhibitor of mRNA transcription prior to detecting or measuring the binding between the ARE and the TZF polypeptide.

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60. The method of claim. 52, wherein the ARE is a class II ARE.

62

61. A method of identifying a compound that mimics the activity of TTP or a TTP-like polypeptide, comprising:

a) contacting a first sample comprising an RNA molecule comprising an ARE with a compound;

b) contacting a second sample comprising an RNA molecule comprising an ARE with the compound and with a saturating amount of a TZF polypeptide consisting essentially of a TTP zinc finger domain or comprising a TTP-like zinc finger domain;

c) detecting or measuring degradation of the RNA molecule or binding of the compound to the ARE in the first sample and in the second sample;

e) comparing the degradation or binding in the first sample to the degradation or binding in the first sample not contacted with the compound; and

f) comparing the degradation or binding in the first sample to the degradation or binding in the second sample, whereby an increase in degradation or binding in the first sample contacted with the compound relative to the sample not contacted with the compound, and lack of an increase in degradation or binding in the second sample contacted with the compound and with the saturating amount of a TZF polypeptide, identifies a compound that mimics the activity of TTP or a TTP-like peptide.

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62. The method of claim. 61, wherein the ARE is a class II ARE.

64

63. A polypeptide consisting essentially of a TTP zinc finger domain or a TTP-like zinc finger domain.

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64. The polypeptide of claim 63, wherein the polypeptide can bind to a class II ARE within an mRNA molecule and stimulate degradation of the mRNA molecule under physiological conditions.

66

65. The polypeptide of claim 63, wherein the TTP-like zinc finger domain is from ERF1, ERF2, or XC3H-4.

67

66. A nucleic acid consisting essentially of a nucleotide sequence that encodes a TTP zinc finger domain or a TTP-like zinc finger domain.

68

67. The nucleic acid of claim 66, wherein the TTP-like zinc finger domain is from ERF1, ERF2, or XC3H-4.

69

68. A vector comprising the nucleic acid of claim 66, wherein the nucleic acid is operably linked to a promoter for transcription of the nucleic acid.